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ORIGINAL ARTICLE

# Immunological profile and dyslipidemia in Egyptian Systemic Lupus Erythematosus patients

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**KEYWORDS**

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**Abstract** *Aim of the work:* To study the relation of the immune profile to dyslipidemia in a cohort of Egyptian Systemic Lupus Erythematosus (SLE) patients.

*Patients and methods:* This study included 221 SLE patients with a disease duration >6 months at study entry. Disease activity was assessed using the SLE Disease Activity Index (SLEDAI) and severity using the Systemic Lupus International Collaborating Clinics/Damage Index (SLICC/DI). Patients were investigated for the anti-nuclear antibody (ANA), anti-double stranded deoxyribonucleic acid (anti-dsDNA), anti-cardiolipin (ACL) antibodies (IgG and IgM), anti Ro (SSA) and anti La (SSB). Dyslipidemia was considered if the high density lipoproteins (HDL), low density lipoproteins (LDL), total cholesterol (TC) or triglycerides (TG) were abnormal.

*Results:* The mean age of the patients was 28.8 ± 7.8 years and the median disease duration was 5 years. The clinical manifestations of the patients were pleurisy (52.9%), pericarditis (24.9%), nephritis (68.3%), CNS lupus (23.1%), vasculitis (14.9%) and musculoskeletal manifestations (57.9%). All patients were on corticosteroids (median dose 35 mg/day; range 5–80 mg/day), while 92 (43.4%) of them received cyclophosphamide during their disease course. The mean SLEDAI was 12.1 ± 7.4 and SLICC/DI was 1.4 ± 1.6. Patients with positive anti-Ro ($n$ = 44; 19.9%) showed statistically significant lower level of HDL ($p$ = 0.01).

*Conclusion:* Positive anti-Ro may be associated with increased incidence of low HDL in lupus patients which in turn may increase the incidence of cardiovascular accidents.


## 1. Introduction

It is proved that Dyslipidemias are being increasingly recognized as an important cause of development of cardiovascular disease (CVD) [1]. An increase of 1% in total cholesterol causes 2% increase in the incidence of ischemic heart disease

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**Table 1** Laboratory data of the Systemic Lupus Erythematosus patients.

| Parameter *n* (%) or median (range) | | SLE patients ($n$ = 221) | |
|---|---|---|---|
| *Immune profile* | | | |
| Positive test | Anti-ANA | 212 | (95.9) |
| | Anti-ds DNA | 150 | (67.9) |
| | ACL | 71 | (32.1) |
| | Anti Ro | 44 | (19.9) |
| | Anti La | 14 | (6.3) |
| *Lipid profile* (mg/dL) | | | |
| High TG | | 79 | (35.7) |
| High cholesterol | | 113 | (51.1) |
| High LDL | | 67 | (30.3) |
| Risk (low) HDL | | 99 | (44.8) |
| Hb (mg/dL) | | 10.5 | (4.5–15.9) |
| TLC ($\times 10^3$) | | 6.9 | (1.4–26.3) |
| PLT ($\times 10^3$) | | 272 | (11–690) |
| ESR (mm/1st h) | | 61.0 | (5–150) |
| ALT (IU/L) | | 21.0 | (6–508) |
| Creatinine (mg/dL) | | 0.75 | (0.2–6.2) |
| Proteinuria (mg/24 h) | | 0.83 | (0–5.9) |
| C3 (mg/dL) | | 80 | (98–178) |
| C4 (mg/dL) | | 15 | (1–130) |

SLE: Systemic Lupus Erythematosus, ANA: antinuclear antibodies, dsDNA: double stranded deoxy-ribonucleic acid, ACL: anti-cardiolipin, Hb hemoglobin, TLC: total leukocytic count, ESR: erythrocyte sedimentation rate, ALT: alanine transaminase, C: complement.

(IHD) [2]. In fact atherosclerotic cardiovascular diseases (ASCVD) are also the leading cause of morbidity and mortality in various autoimmune diseases, mainly rheumatoid arthritis, antiphospholipid syndrome (APS) and Systemic Lupus Erythematosus (SLE) [3,4]. SLE is a complex autoimmune disease characterized by immune disturbances and involvement of multiple systems and organs. Dyslipidemia in Egyptian SLE patients was reported to be associated with decreased quality of life and damage accrual with an increased risk of atherosclerosis [5].

Although the survival of SLE patients has improved during the past three decades; however, they still die at a rate that is three times higher than that of the general population [6] while early deaths could be explained by active disease and infection, late deaths (patients aged >40 years) are mainly attributed to CVD [7,8]. Recent advances in the etiology of accelerated atherosclerosis in SLE have stressed the relevance of the interplay between lupus-specific inflammatory factors and traditional cardiac risk factors in causing increased endothelial damage [9].

The presence of autoantibodies is a cardinal feature of SLE [10]. Quantification on the serum levels and/or prevalence of one or more autoantibodies recently demonstrated their significance in the disease pathology [11]. Anti-double stranded deoxy-ribonucleic acid (anti-dsDNA) antibodies were significantly associated with lupus nephritis (LN) [12,13] and both anti-Ro and anti-La antibodies were strongly associated with skin manifestations [14] in SLE patients.

Thus the aim of the current study was to investigate the relation of SLE associated autoantibodies and dyslipidemia in a cohort of Egyptian patients.

## 2. Patients and methods

The study, descriptive and cross-sectional, included 221 Egyptian SLE patients fulfilling the updated ACR revised criteria for the classification of SLE [15]. All the patients had the disease duration more than 6 months at study entry. The patients were attending the Rheumatology department of Cairo University Hospitals during 2014.

All Patients were subjected to full history taking, thorough clinical examination, laboratory and relevant radiological investigations were performed for all the patients. Disease activity was assessed using the SLE Disease Activity Index (SLEDAI) [16] and severity using the Systemic Lupus International Collaborating Clinics/Damage Index (SLICC/DI) [17].

The clinical manifestations are described based on the SLEDAI and SLICC items. The study has been approved by local ethics committee and it conforms to the standards currently applied in Cairo University Teaching Hospitals. Patients gave an informed consent before being enrolled in the study.

The information collected included the history of smoking, alcohol intake, presence of diabetes mellitus, and dose of steroid. A detailed dietary history was also included.

For all patients, serological examinations were performed including the following: (A) Immune profile: Anti-nuclear antibody and anti-ds DNA tests were carried out by indirect Immunofluorescence (IIF) technique; anti-cardiolipin antibodies (ACL) (IgG and IgM) and anti-Ro (SSA) and Anti-La (SSB) were detected by ELISA. (B) Lipid profile: high density lipoproteins (HDL), low density lipoproteins (LDL), total cholesterol (TC) and triglycerides (TG) were checked. A diagnosis of dyslipidemia was given if the patient had (a) hypercholesterolemia, defined as taking lipid-lowering medication as a surrogate, or having a fasting plasma TC $\geqslant$ 200 mg/dL (b) HDL $\leqslant$ 40 mg/dL, (c) hypertriglyceridemia (TG $\geqslant$ 150 mg/dL) or (d) elevated LDL ($\geqslant$100 mg/dL) [18].

*Statistical analysis*: Data were statistically described in terms of range, mean ± standard deviation (SD), median, frequencies (number of cases) and percentages when appropriate. Comparison of quantitative variables between the study groups was done using Mann–Whitney U-test for independent samples. For comparing categorical data, Chi-square (v2) test was performed. Exact test was used instead when the expected frequency is less than 5. A probability value ($p$ value) less than 0.05 was considered statistically significant. All statistical calculations were done using computer programs Microsoft Excel 2003 (Microsoft Corporation, NY, USA) and SPSS (Statistical Package for the Social Science; SPSS Inc., Chicago, IL, USA) version 15 for Microsoft Windows.

## 3. Results

This study included 221 SLE patients, 212 (95.9%) females and 9 males (4.1%). Their ages ranged from 17 to 54 years with a mean of 28.8 ± 7.8 years. Their disease duration ranged from 0.5 to 20 years with a median of 5 years. The main clinical presentation of our patients included pleurisy (52.9%), pericarditis (24.9%), nephritis (68.3%), CNS lupus (23.1%), vasculitis (14.9%), and musculoskeletal manifestations (57.9%). None of our patients had history of smoking, alcohol intake or presence of diabetes mellitus. All of our patients were on corticosteroids, their doses ranged from 5 to 80 mg/day